LIPOPROTEIN PROFILE CHANGES DURING THE PROCESS OF LABOUR AND TWENTY-FOUR HOURS OF POST PARTUM PERIOD

THESIS

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MASTER OF SURGERY

(OBSTETRICS & GYNAECOLOGY)

BUNDELKHAND UNIVERSITY
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1998 ABHILASHA CHAUHAN

C E R T I F I C A T E

This is to certify that the work entitled "STUDY OF LIPOPROTEIN PROFILE CHANGES DURING THE PROCESS OF LABOUR AND TWENTY FOUR HOURS OF POST PARTUM PERIOD", which is being submitted as a thesis for M.S. (Obstetrics & Gynaecology) Examination, 1998, Bundelkhand University by DR. ABHILASHA CHAUHAN, has been carried out in the department of Obstetrics and Gynaecology, M.L.B. Medical College, Jhansi.

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(Abhilasha Chauhan)

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I N T R O D U C T I O N

INTRODUCTION

Pregnancy is accompanied by profound hormonal alterations and these endocrinal changes during gestation have been proved to be the causative factor altering the lipid metabolism.

In early weeks of pregnancy the corpus luteum serves as a source of hormones soon the action is taken over by placenta which serves as the main endocrine organ bringing about continued and higher production of steroid and other hormones. Besides the anterior pituitary, adrenal cortex also have an important role to play in the extrapolation of hormones, which support pregnancy.

The oestrogen level during pregnancy increases progressively to reach maximum at term then values of oestrogen falls within 3 days and reaches at the basal level by 7th post partum day. Oestrogen causes increase in HDL cholesterol while LDL decreases. Biosynthesis of VLDL is enhanced while triglyceride lipase activity reduced.

Progesterone brings about a decrease in HDL-c and increase in LDL-c, it reduces hepatic triglyceride lipase activity.

The hyperlipidemia of pregnancy is potentially significant from several stand points. (1) The rise in plasma triglyceride may enhance the availability of essential and non-essential triglyceride fatty acids for placental transfer to fetus. (2) The cholesterol rise

may increase the supply of cholesterol needed for placental progesterone synthesis and transplacental cholesterol transfer to the fetus. (3) The plasma triglyceride elevation may be the barometer of a general metabolic adaptation by the mother to augment nutrient flow to the fetus. (4) The hyperlipidemia may stress maternal lipid homeostasis to an extent that subclinical or mild hyperlipidemia becomes clinically detectable anatogous to the prediabetes recognised in a women when she develops gestational diabetes. (5) The hyperlipidemia of pregnancy could itself function as an atherosclerosis risk factor.

The pattern of change in level of serum cholesterol and triglyceride during pregnancy is as follows. The serum cholesterol and triglyceride levels steadily increase starting from second trimester upto term and attain a peak just prior to the onset of labour and then abruptly falls with the expulsion of placenta, but not reaching the pregestational levels. In post partum period these fall gradually.

The abrupt fall in lipoprotein levels that occurs immediately after delivery also raises some queries. How this raised lipoprotein disappears all of sudden ? Where does this lipoprotein go? Is it shifted to fetal circulation during labour ? or is it shifted from maternal circulation to extravascular/subendothelial compartment ?

Although studies are available for the antepartum and postpartum phase,not much has been done during intrapartum phase. Hence a study had to made regarding the changes in lipoprotein profile during actual process of labour. Thus in this study it was observed whether there is any relation of serum lipoprotein profile to the process of labour whether preterm, term pregnancy - induced or spontaneous : normal or abnormal, emergency or elective caesarean section.

AIMS AND OBJECTIVES

1. To study the lipoprotein profile changes during various stages of labour and within 24 hours of postpartum perio
2. To study the lipoprotein profile changes, in relation t mode of delivery e.g. vaginal delivery, induced vaginal delivery, elective or emergency caesarean section.
3. To study the variation in lipoprotein fractions in umbilical cord blood of newborn with respect to mode of delivery. of mother.
4. To study the effect of parity on lipoprotein fractions during various stages of labour and postpartum period.
5. To study the role of diet (vegetarian Vs nonvegetarian) on lipoprotein fractions during intrapartum and postpartum phase of mother and umbilical cord blood of newborn baby.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

In pregnancy there is re-adjustment of hormones on the part of the mother, almost every endocrine tissue participates in the adaptive changes, that maintains the metabolic state of female during normal pregnancy.

The occurrence of hyperlipidemia during normal pregnancy was known as early as the 1845 (Baqueral and Rodier). Milky appearance of sera of pregnant woman, was due to presence of fat, was showed by Virchow (1847).

The first chemical study was undertaken in 1911 when Chaufford demonstrated an increase of blood cholesterol during normal pregnancy. At the same time Hermann and Neumann studied the lipid particles in whole blood and reported an increase in cholesterol during pregnancy. They concluded that during first 6-7 months, the serum cholesterol might be increased and that during the last two months the increase in serum cholesterol was a rule.

Eminent investigators (Boyd, 1934; Dieckmann and Wegner, 1934; Schwartz et al, 1940; Peters et al, 1947; Russ et al, 1954; and Jacina et al, 1961) observed in their works that normal status is changed and there was an increase in serum cholesterol phospholipids and neutral fat which progressed towards term and decreased after delivery.

Tyler and Underhill (1925) determined the whole blood cholesterol in normal pregnant woman, in each month

of pregnancy and reported that cholesterol increases gradually untill term, when it is roughly one third higher than at three months.

LABOUR

Series of events take place in the genital organs in an efforts to expel the viable products of conception out of the womb through the vagina into outer world is called labour. Parturition is the process of giving birth. Delivery is the expulsion or extraction of a viable fetus out of the womb. It is not synonymous with labour; delivery can take place without labour as in elective caesarean section. Delivery may be vaginal either spontaneous or aided or it may be abdominal.

Normal Labour

Labour is called normal if it fulfills the following criteria :

1. Spontaneous in onset and at term.
2. With vertex presentation.
3. Without undue prolongation.
4. Natural termination with minimal aids.
5. Without having any complications affecting the health of mother and/or the baby.

Stages of Labour

Conventionally, events of labour are divided into three stages as below :

First stage

It starts from the onset of true labour pain and ends with full dilatation of the cervix. It is, in other words, the 'cervical stage' of labour. Its average duration is 12 hours in primigravida and 6 hours in multigravida.

Second stage

It starts from the full dilatation of the cervix and ends with expulsion of the foetus from the birth canal. Its average duration is 2 hours in primigravida and 30 minutes in multigravida.

Third stage

It begins after expulsion of foetus and ends with expulsion of the placenta and membrane (after births). Its average duration is about 15 minutes in both primigravida and multigravida. The duration is, however, reduced to 5 minutes in the active management.

Fourth stage

It is a stage of observation for at least one hour after expulsion after birth. During this period, general condition of the patients and the behaviour of the uterus are to be carefully watched.

MECHANISM OF ONSET OF LABOUR

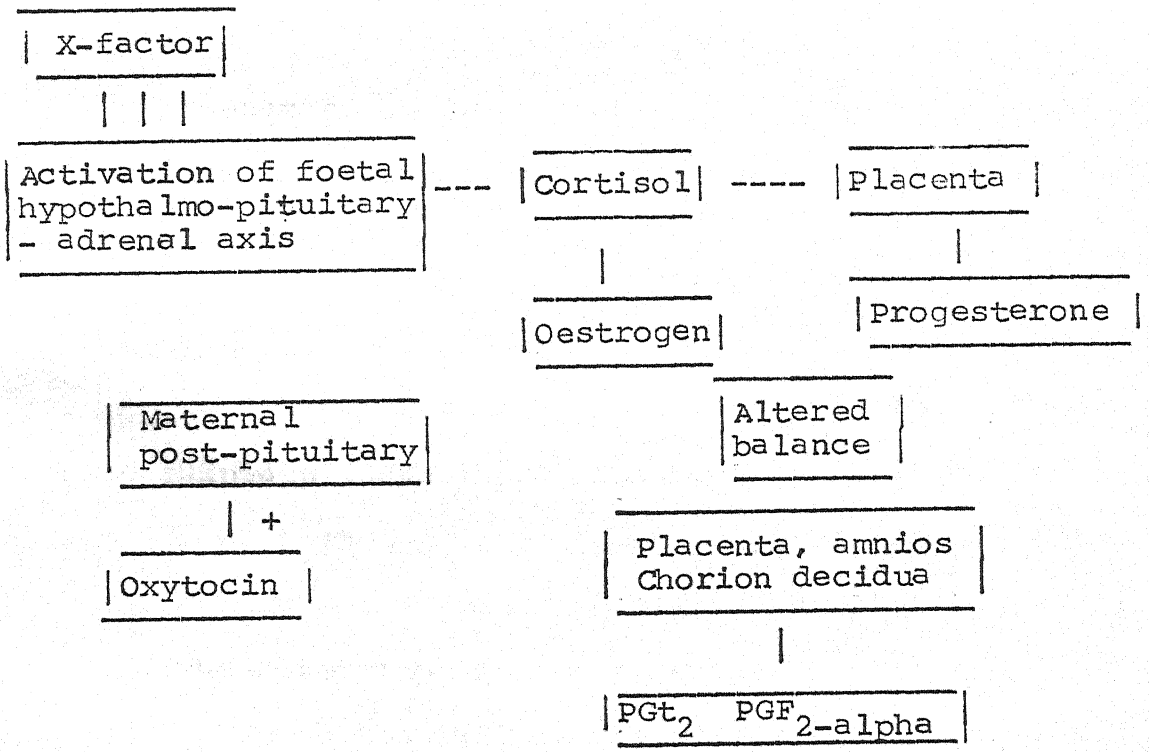
The precise mechanism of initiation of labour is still obscure. Advancement of chemico-hormonal technology and inferences obtained from animal experiments

put forth the following hypotheses.

1. Uterine Distension

Stretching effect on the myometrium by the growing size of the foetus and liqour amnii can be explain the onset of labour at least in twins or hydro-amnios. However, optimal distension theory fails to account for the otherwise causeless preterm labour.

Possible mechanism of initiation of labour



2. Foetoplacental Contribution

It has been postulated that due to unknown factors feotal pituitary is stimulated prior to onset of

labour ---- | release of P.C.T.H. ---- Stimulates foetal adrenals ------ | cortisol secretion ---- accelerated production of oestrogen and prostaglandins from the placenta. The probable modes of action of oestrogen are:

1. Increases the release of oxytocin from maternal pituitary.
2. Promotes the synthesis of receptors for oxytocin in the myometrium and decidua.
3. Accelerates lysosomal disintegration inside the decidual cells thus increased prostaglandin synthesis.
4. Stimulates the synthesis of myometrial contractile protein actomyosin through activation of adenosine triphosphatase.
5. Inreases the excitability of the myometrial cell membranes.

3. Progesterone

Increased foetal production of dehydro epiandrosterone sulphate (DHEAS) and cortisol may inhibit the conversion of foetal preg henabine to progesterone, thereby altering the oestrogen-progesterone ratio. It is probably the alteration in the oestrogen : progesterone ratio rather than the fall in the absolute concentration of progresterone which is linked with prostaglandin synthesis.

4. Prostaglandins

Prostaglandins have attracted much attention in recent years as a factor to initiate and maintain labour.

The major sites of synthesis of prostaglandins are placenta, membranes, decidua cells and myometrium. Synthesis is trigerred by rise in oestrogen level altered oestrogen - progesterone balance, mechanical streching in late pregnancy, increase in oxytocin receptors, infection, separation or rupture of membranes.

The prostaglandin synthesis reaches a peak during the birth of placenta probably contributing to its expulsion and to the control of postpartum haemorrhage.

5. Oxytocin

There is no conclusive proof that oxytocin level is increased prior to labour, there is, however, increase in oxytocin receptors especially in decidua vera which in turn stimulates prostaglandin synthesis. Oxytocin level reaches the maximum at the moment of birth and is caused by a reflex release due to vaginal distension.

6. Nervous Factor

Although labour may start in denervated uterus, labour may also be initiated through nerve pathways. Both alpha and beta receptors are present in the myometrium and beta receptors to function predominantly. The contractile response is initiated through the alpha receptors of the post ganglionic nerve fibres in and around the cervix and lower part of uterus. This based on the observations of the onset of labour following stripping or low rupture of the membranes.

7. Cortisol

The pituitary adrenal axis of the fetus gives the signal for the initiation of labour. Increase in cortisol levels occurs which raises oestrogen levels. High cortisol levels have been observed in patients of spontaneous labour as compared to caesarean section. These are also increased in foetal and maternal stress during active labour. Cortisol might compete with progesterone receptors in myometrium by binding with the progesterone carrier protein.

An encephaly, postmaturity and adrenal hypoplasia is associated with prolonged gestation due to reduced cortisol levels due to reduced foetal adrenal function.

LIPOPROTEIN PROFILE

The lipids are a heterogenous group of compounds related, either actually, to the fatty acids. They have the common property of being insoluble in water and soluble in non polar solvents. Lipids are important dietary constituents not only because of their high energy value but also because of the fat soluble vitamins and the essential fatty acids contained in the fat of natural foods.

The following classification of lipids is modified from Bloor.

A. Simple lipids : Esters of fatty acids with various alcohols.

1. Fat : Esters of fatty acids with glycerol, a fat in lipid state is called oil.

2. Waxes : Esters of fatty acids with higher molecular weight monohydric alcohols.

B. Complex lipids : Esters of fatty acids containing groups in addition to an alcohol and fatty acids.

1. Phospholipids : Lipids containing, in addition to fatty acids and an alcohol, a phosphoric acid residue, they have nitrogen containing bases and other substituents.

2. Glycolipids : Lipids containing a fatty acid, sphinage sine and carbohydrates.

3. Other complex lipids - Lipids such as sulpholipids and aminolipids. Lipoproteins may also placed in this category.

4. Precursor and derived lipids - these includes hormones and vitamines.

Cholesterol

It is probably best known steroid because of its association with atherosclerosis. Also this is the precursor of large number of steroids i.e. bile acids, adrenal hormones, sex hormone and Vitamin D etc.

It is widely distributed in all cells of the body. It is the major constituents of the plasma membrane and plasma lipoprotein. It is typically product of animal metabolism and so occurs in food of animal origin e.g. egg. yolk, liver meat etc. The normal level of serum cholesterol ranges from 150-250 mg/dl.

Triglycerides

It forms main bulk of dietary lipids. It is neutral fat. Normal values ranges from 80-240 mg/dl.

Lipoproteins

Lipids are present in plasma in the form of lipoproteins mainly which serve the two important functions - first is transport of triglycerol and another is the transport of cholesterol and its esters. The protein part of lipoprotein is called apoprotein. The lipid content of lipoprotein decreases the density of lipoproteins due to difference in nature and molecular weight of lipid and protein portion of several lipoproteins give them different densities and permit their further separation is centrifuge.

Thus, on the basis of different densities, lipoproteins can be of following types :

a. Chylomicrons

These are formed only by the lymphatic system draining the intestine. The levels fluctuates with the load of triacylglycerol absorbed. They are cleared rapidly from the circulation within one hour.

b. VLDL or Prebeta lipoprotein

These consist mainly of glycerides that are endogenous. It is formed by the hepatic parenchymal cells. Its formation is constant and even occurs in fasting state. It acts as a vehicle of transport of triglycerol from liver to the extrahepatic tissues.

Half life of VLDL is 2 hours and virtually all of the VLDL is converted into LDL metabolically. The level of VLDL between 20-29 years is about 25 mg/dl and between 30-39 years it is 35 mg/dl.

c. Beta-lipoprotein : Low density lipoprotein (LDL)

It is mainly formed by breakdown of VLDL in circulation. They are divided in LDL_1 and LDL_2 types depending upon densities. Cholesterol concentration in plasma is a reflection of the LDL concentration.

Half life of LDL is about 4 days, range of LDL level in circulation is between 170-190 mg/dl.

d. High Density Lipoprotein (HDL)

Also called alpha lipoproteins. These are much smaller than other lipoprotein. These are separated between densities of 1.063 - 1.210 and contain about 50% protein. It helps in destruction of IDL and LDL. It is formed in liver mainly and to a lesser extent also in intestinal epithelium. HDL has a protective role in coronary artery disease. High level of HDL in blood indicates less chances of atherosclerosis.

Normal range of HDL in blood is 75 mg/dl in age group of 20-29 years and 80 mg/dl in the age group of 30 to 39 years.

Plase and Tompkins (1923) have also given figures for the blood and lipids particularly cholesterol during pregnancy. These figures indicate a gradual rise from 4th

month to term.

Gardner and Gainsborough (1929) studied the ratio of cholesterol to cholesterol esters and reported an increase in cholesterol with a decrease in cholesterol ester till the 30th week of pregnancy. In their series reversal of the relationship occurs so that at parturition approximately a normal relationship exists again.

Kaufmann and Mihlback (1933) did not notice these fluctuations but they reported little variations from the second month of gestation to term.

According to Dieckman and Weigner (1934) total cholesterol increases to 23% above the first trimester levels and which decreased to 27% at the 8th postpartum week from the values at term.

On the basis of study of primigravida, Oliver and Boyd (1935) stated that there is significant rise in plasma ester and total cholesterol between 31st and 33rd week of pregnancy. By the 20th week of postpartum period values have decreased but still higher than the levels at 12th week of pregnancy.

After a serial estimation of plasma esterified fatty acids on puerperal patients following delivery and through fifth postpartum day, Dannenburg et al (1962) concluded that mean puerperal values fro total carboxyl esters were higher than the nonpregnant class. This was statistically significant.

All fractions of esterified fatty acids except cholesterol esters showed a slight increase within 24 hours of delivery and then declined subsequently. These observations were similar with study of Boyd (1935) and Schwarz (1940) but according to Peters (1951), Watson (1957), there is a drop in plasma lipids following delivery.

Two phase study to define and detect changes in total lipids and their fractions during various periods of gestation was done by Mullick and Bagga (1964), according to this study all lipid fractions show a gradual and persistent rise throughout pregnancy.

Bhattacharya (1969) on the basis of their study, concluded that although cholesterol levels were slightly higher in toxaemia group. The cholesterol metabolism seemed to be similar in normal and toxaemia of pregnancy.

Potter and Nistel (1979) studied lipoprotein profile during pregnancy and puerperium in a group of 43 women. The plasma cholesterol concentration was raised by about 50% and major rise was in second trimester. The plasma triglyceride concentration was raised 3 fold and peak was found during third trimester.

Sita Devi and Patrudu et al (1981) longitudinal study of serum lipoprotein in normal pregnancy and puerperium, estimated lipid profile in 23 pregnant women in all three trimesters and in post natal period. Thirty three normal healthy nonpregnant women of the same age group fromed the control. A progressive rise was observed in serum triglyceride and cholesterol reaching the peak in the

3rd trimester and definite fall in post natal period as compared to antenatal period among hyperlipemias, the major one was type IV in the 1st trimester, type IIb and type IV in 2nd trimester and type IIb in 3rd trimester, and type IV in the post natal period. It was also noted that those who had an abnormal lipid profile in the 1st trimester continued to have abnormal lipid profile in the post natal period.

Darmandy and Postle (1982) measured serum lipid concentration in a group of normal women from before conception to throughout gestation and untill at least 40 weeks after delivery. The effect of lactation was also studied. The primary change in lipoprotein metabolism during pregnancy, appears to concerned with VLDL which are elevated, the rate of secretion depending upon the lipoprotein lipase activity. After delivery the elevated serum triglyceride decreases rapidly and significant utilisation of serum triglyceride in lactating women could be caused by the tissue specific direction of VLDL towards the mammary gland for milk synthesis.

Fahraeus Lars et al (1985) studied levels of plasma lipoprotein fractions in 19 women at exact gestational ages. The HDL levels elevated in the 14th week and showed a maximum rise of 41% in 28th week of pregnancy. The LDL decreased in early pregnancy but then increased continuously. The VLDL concentration showed an increase from 14 to 36 week. During lactational period, 8 weeks after delivery the LDL concentration remained elevated whereas

the other lipoprotein had returned to pregnancy level.

Erkkola and Viikari et al (1986) investigated serum lipid and lipoprotein fractions one day after delivery, 3 months later in lactating and non lactating mothers and 12 months later after initiation of menstruation in a group of 62 women. 29 of which formed a truly longitudinal group. STC decreased significantly during the following 9 months. LDL & HDL showed also a significant decrease within the postpartum period. Serum triglycerides decreased with 3 months after delivery but no more significantly lower. In lactating mothers HDL : STC ratio was higher than in non lactating women. During luteal phase STC and LDL were lower and HDL : STC ratio was higher than earlier during the menstrual cycle. Pregnancy related changes in lipid metabolism did not wane within 3 months after delivery.

Arora and Vinita (1987), in their study, showed STC level of 166.17±24.97 mg/dl in 1st trimester while reached to a peak of of 263.44±39.8 mg/dl during labour. This decreased in the post partum period reaching 190.5± 36.94 mg/dl at one month of post partum period.

Herrera et al (1988) did their work on mechanism involved in maternal hypertriglyceridemia in late normal pregnancy and the physiologic significance reviewed as a model of the effects of sex hormones steroids in lipoprotein metabolism. They concluded that changes in magnitude and even direction of lipoprotein lipase activity in different tissues during gestation contribute not only to be metabolic fate of circulating triglycerides. These

dynamic and metabolic adaptations seen in the mother may directly modify her lipoprotein profile. Under pathological conditions the alterations may be permanently maintained thereby increasing the risk for the development of cardiovascular disease.

Valimaki et al (1990) have studied the serum lipid and lipoproteins in a alcoholic women during pregnancy, and concluded that alcohol abuse clearly reduced the normal increase in total cholesterol and LDL cholesterol during 24 to 40 weeks. HDL-3 was raised and normal increase in VLDL was accentuated in these women.

Arora and Neeta et al (1993) studied the changes in lipoprotein profile in normal pregnancy and artificial termination of pregnancy (Elective/emergency LSCS). They observed that there was rising trend in lipoprotein profile with a peak during labour followed by fall in the post partum period, both in normal as well as artificial termination of pregnancy.

Mazurkiewicz, Walts et al (1994) in new study on serum lipids, lipoproteins and apolipoproteins in pregnant non-diabetic women reported that pregnant women had significant higher concentration of STG, STC, HDL, LDL and apolipoproteins A & B, also that the ratio of STC : HDL, cholesterol was not significantly different.

Udoh and Ham et al (1994) in their study on normal pregnant women observed a progressive increase in the serum total cholesterol and HDL-c throughout pregnancy and decrease in their levels after delivery. In their study on 49 normal

pregnant women STC rose from 154.91±15.39 mg/dl at after one month of delivery. This represents a 39±11 percent increase in STC at 9 months from the 3 months levels. HDL-c showed an increase of 35±10 percent at 9 months level from 3 months level. The most significant months to months increase was recorded between the 6th and 7th month for both STC and HDL-c.

Chaing and Yang et al (1995) in their study of lipid profile in normal pregnancy found that serum total cholesterol, triglycerides, LDL-c were significantly elevated during second and third trimester of pregnancy but dropped sharply after delivery.

LIPID PROFILE IN UMBILICAL CORD BLOOD

So many studies have shown that in maternal blood the concentration of cholesterol and phospholipids is greater than normal while in umbilical cord blood at the time of birth is significantly reduced.

Boyd and Wilson (1934) studied exchange of lipid in the umbilical circulation at birth. They took samples of venous blood from the maternal end of cord with the placenta still attached in utero. This represented the venous blood entering the fetus. The concentration of uterus was assumed to have little effect upon the lipid content of venous blood, on assumption which was substantiated in past by the finding of similar results in cases of caesarean section in which the uterus was not contracted.

It was later found that lipid concentration of venous blood slowly increases after the cord is clamped. They concluded that phospholipids, free cholesterol and cholesterol esters are added to umbilical blood between the time of delivery and the time of placenta separation. Neutral fats may be either removed or added to umbilical blood by the placenta.

Sa dowsky et al (1947) observed the cord and maternal cholesterol values in babies delivered normally. Mean cord blood cholesterol values were 107 mg/dl which were higher than observed by earlier workers while the maternal mean blood cholesterol values were 262 mg/dl which were comparable to values obtained earlier.

Brown et al (1959) studied maternal serum during 1st stage of labour and cord blood samples collected just after birth. The mother had normal full term delivery. The maternal values were 1104±172 mg/dl, 257±71 mg/dl, 847±176 mg/dl and 273±52 mg/dl for total lipids, lipoprotein lipids, beta lipoprotein and cholesterol respectively. The values in cord blood samples were 371±75 mg/dl, 147±40 mg/dl, 224±41 mg/dl and 82±17 mg/dl respectively.

Brady and Carlon (1962) found that the concentration of serum triglycerides is quite low in the cord blood and newborn. This has been confirmed by the Kaplan and Lee (1965) who had the same observations.

Konttinen et al (1964) studied serum lipids in normal pregnancy and pre-eclampsia and also umbilical cord

blood of groups. They concluded that clord blood samples of both the groups showed low levels of all the lipids studied and no difference was detectable between the two groups. The mean total cholesterol was about 80 mg/dl with a high content carried in alpha fractions. The serum triglycerides values were only about 1/8th of the values seen in their mothers with no individual correlation between mother and child.

Ortega, Gasper et al (1966) in their study on influence of maternal serum lipids and maternal diet during the third trimester of pregnancy and mubilical cord blood lipids in two population of Spanish newborns noted that a significant correlation was found to exist between maternal cholesterol concentration and those of newborn infant. A correlation was also found between maternal cholesterol levels and infant HDL-c and LDL-c levels. Further a positive correlation wasc seen between maternal LDL-c and infant cholesterol and LDL-c. The relationship between maternal cholesterol and cord blood cholesterol was independent of participants' dietary anthropometric and personal data. 3.1% of neonates showed total cord blood cholesterol concentration of 799.9 mg/dl. The mother of these children showed the strongest concentration of cholesterol and LDL-c in the 3rd trimester of pregnancy, the shortest pregnancies and the smallest newborns of all subjects. Negative correlations were found between birth weight and cord blood cholesterol levels at LDL-c.

Pontis et al (1979) studied antepartum and post partum lipoprotein levels in parturiting women and in umbilical cord blood of their newborns. The average values reported in umbilical cord blood were far below than that of maternal blood. The difference that exists between mother and baby in this respect varies from case to case and values prevailing in one seems to be entirely independent of these in other i.e. concentration of cholesterol is never same in mother and foetus. The difference has no constant or characteristic pattern.

Arora and Kavita et al (1989) in their study of changes in lipoprotein profile in normal pregnancy and toxaemia of pregnancy during antepartum and post partum periods and in umbilical cord blood of their newborns, found that STC, HDLc, STG, VLDL and LDL values of cord blood were very low in comparison to intrapartum values of mothers. Difference in the levels of HDL and STG in normal pregnancy and toxaemia of pregnancy were found to be statistically significant.

Heary and Kilby et al (1994) in their study on foetal and maternal lipoprotein metabolism in human pregnancy determined the concentration and composition of lipid and apolipoprotein in maternal venous and umbilical arterial and venous blood. The objective of the study was to establish whether the placenta has a role in feto-meternal cholesterol metabolism through either synthesis or transplacental cholesterol flux. Study showed that pregnant women had raised

levels of all lipids and lipoprotein fractions as compared with control subjects in both umbilical venous and arterial blood content. Ratio of VLDL, LDL, cholesterol esters and triglycerides were lower than in maternal blood, but HDL-c levels were similar. There was no umbilical arterio-venous difference in lipoprotein concentration or composition. They suggests that cholesterol synthesis or free cholesterol diffusion do not occur in the placenta.

EFFECT OF DIET ON SERUM LIPOPROTEIN PROFILE

A study on cholesterol metabolism was done in 1929 by Gardener and Gainsbrough and concluded that during period of fasting, cholesterol content of plasma varies markably in different healthy persons but is fairly constant in subjects A single meal does not cause any changes but prolonged diets high or low in sterol will cause variation in cholesterol, the free cholesterol remains fairly constant but cholesterol esters show greatest change.

According to Mullick and Bagga (1964) in healthy females serum lipids and its fractions vary with the nutritional status which is itself dependent upon the socio-economic conditions of individual. Values for high income groups are close to those reported by Boyd. In pregnant fenales the increase in total serum lipids occurring in first 8 weeks of first trimester was more marked in vegetarians than in the nonvegetarians. STC, ester cholesterol and free cholesterol showed the reverse trend. In the 2nd trimester, this difference in the serum total lipids was

narrowed. In 3rd trimester, there was no difference in serum total lipids between vegetarians and nonvegetarians, but there was now a slight increase for the non-vegetarians. Thus diet has no significant influence in lipid synthesis in the later period of pregnancy.

Geen Gun (1966) determined total cholesterol serially in a group of young women before and during pregnancy while they consumed their usual diet or a fat modified diet known to have a hypocholesterolemic effect. During 1st trimester of pregnancy there was a slight but definite fall in serum cholesterol level. After the 1st trimester serum cholesterol level increases gradually to peak at or near term. These changes occur both in normal and hypercholesterolemic females and is not affected by fat modified diets.

Hansen and coworkers studied 80 pregnant women and found no significant correlation between mother's intake of calories, proteins, fat and fatty acids to serum cholesterol and fatty acids levels during 3rd trimester.

Arora and Vinita (1987) studied the influence of dietary fat on STC levels during antepartum, intrapartum and post partum periods of pregnancy and in the cord blood of newborns, and concluded that the levels of STC were higher in subjects taking high fat diet and lower in those taking low and normal fat diet with advancement of pregnancy during labour, after delivery and in late postpartum period values were not significant. However, the cord blood STC values in relation to fat diet of mother in 3rd trimester were highly significant.

HORMONAL CHANGES DURING PREGNANCY, LABOUR AND POSTPARTUM PERIOD AND ROLE OF HORMONES IN LIPORROTEIN RROFILE CHANGES

The endocrine plays very important role in physiology of reproduction. i.e. following conception transfer of function of pituitary - ovarian axis to placenta which acts temporarily as a new dendocrine organ. During pregnancy, there is physiological alteration of endocrine glands, namely the pituitary, thyroid, parathyroid, adrenal and pancreas to maintain the conception, to promote growth and to control labour. Hormonal influence during puerperium is necessary for maintenance of lactation.

HORMONES OF PLACENTA:

At 6 - 8 weeks there is transfer of function of corpus luteum to the placenta which acts temporarily as a new endocrine organ. It produces variety of hormones of which steroids and protein hormones are important

a. Steroid Hormones:

   i. Oestrogens

   ii. Progesterone

b. Protein hormones:

   - Human chorionic gonadotrophin (HCG)

   - Human placental lactogen (HPL).

1. OESTROGENS

Oestriol is the main pregnancy oestrogen accounting for 80-90% of the total oestrogen formed. The oestrogen levels during pregnancy increases progressively to reach a level of approximately 150 mg/ml at terin (Ronald K. Kalkhoff et al , 1978), a level almost 16 times higher than the values at 8 weeks (Ddesoye et al, 1986).

Oestrogen levels fall significantly within 3 days and reach a basal level by 7th postpartum day, rising again in nonlactating by the 14th day.

Estrogens cause an increase in HDL-c while LDL is decreased. The biosynthesis of VLDL is enhanced but the triglyceride lipase activity reduced.

2. PROGESTERONE

After the first trimester, the placenta becomes capable of producing sufficient progesterone to maintain gestation, the levels of which in maternal plasma increases progressively with gestation. Cholesterol derived from maternal blood is the main substrate for the trophoblastic synthesis of progesterone. The levels of the hormone secreted by the placenta approximates 250 mg/day, the levels at term being 7 times the value at 8 weeks.

Progesterone levels fall significantly within 3 days reaching basal levels by 7th day in lactating mothers.

The oestrogen to progesterone ratio is also increased from 0.08 in the first trimester to a value of 0.232 at about 35 weeks. Thereafter it decreased to 0.187 at 38 weeks. No association has been found between lipid and lipoprotein levels to the oestrogen to progesterone ratio, except that the fall of ratio was parallel to the fall in LDL levels immediately before term (Desoye et al, 1986).

Individually progesterone brings about a decrease in HDL cholesterol and an increase in LDL-c . It induces hepatic triglyceride lipase activity. Increased degradation of VLDL and/or IDL resulting in high plasma LDL-c levels.

3. HUMAN PLACENTAL LACTOGEN

A polypeptide hormone secreted by the placenta gradually increases and eventually reaches a maximum of 5-8 ugm/ml at term. Maternal concentrations promptly return to undetectable levels within 24 hours.

The levels of HPL clearly parallel the time course o of the lipids changes during pregnancy. It has lipolytic activity thus releasing FFA probably by activation of hormone sensitiva lipase. This may occur for foetal requirements in the second half of pregnancy during which the mass of maternal adipocytes are reduced and the foetus gains weight. The portion of free fatty acids not utilised by foetus is incorporated into STG and VLDL in maternal liver (Desoye et al, 1987).

4. HUMAN CHORTIONIC GONADOTROPIN(HCG)

Concentration of HCG rises to peak values by 8-12 weeks of gestation. Thereafter there is a decrease in HCG levels to a plateau that is maintained throughout the remainder of pregnancy. It becomes undetectable in urine by 7-10 days postpartum. Free cholesterol was inversely related to HCG levels whereas triglyceride concentration resembled those to insulin.

B. ANTERIOR PITUITARY HORMONES

1. HUMAN GROWTH HORMONE (HGH)

Basal levels of growth hormone are low during early pregnancy and do not change remarkably with advancing gestation. Pituitary somatotrphic hormone or growth hormone raises the blood lipid levels. Slow rise in HGH occurs during postpartum phase.

2. PROLACTIN

Concentration of serum prolactin in pregnancy begin to increase approximately 30 days after the mid menstrual cycle peak of lutienising hormone. Rising prolactin levels continue to increase to reach peak levels at term. Serum prolactin declinea rapidly after parturition, if the woman does not breast feed. However, prolactin levels increase sharply with breast feeding episodes, they then decrease to non pregnant values after several months of lactation.

C. THYROID HORMONES

1. THYROXIN

Like oextrogens it depresses the blood lipid levels. Patterson, Hund and Nicodeus (1938) believed that hypercholesterolemia of pregnancy is due to subclinical hypothyroidism.

Lister (1955) and Russell (1956) found that protein bound iodine and serum precipttate iodine are elevated as early as second month of pregnancy. These levels have been found to reach as high as those seen in individual with overt hyperthyroidism.

Strisower (1958) found that the thyroid hormone depresses serum lipid partition but during pregnancy the tissue become more refractory to the effect of thyroxin.

D. ADRENAL HORMONES

GLUCOCORTICOIDS

Cortisol metabolism is significantly altered during pregnancy, the maternal plasma levels rising progressively throughout gestation. The plasma levels of transcortin also rises progressively to peak in third trimester. The increased transcortin levels are due to increased oestrogen concentration. The maternal tissues are exposed to an average daily concertration of cortisol that is more than twice normal. Cortisone, increases the cholesterol and its ester level and thus is achieved at the cost of neutral fats (Jailer et al, 1957).

E. PANCREATIC HORMONES

INSULIN

The basal levels of insulin tend to become progressively higher as term gestation is approached. Also a much greater amount of insulin is released in response to glucose stimulation. However, during pregnancy a state of insulin resistance exists. Insulin has a pronounced antilipolytic effect and antagonises the lipolytic effect of hormones mainly by inhibiting the hormone sensitive lipase in the adipose tissue. Thus it reduces the release and only of free fatty acids but of glycerol as well.

MATERIAL AND METHODS

MATERIAL AND METHODS

The present work entitled "Study of lipoprotein profile changes during the process of labour and 24 hours of post partum period" was carried out in the department of Obstetrics & Gynaecology and Lipid Research Laboratory, Department of Medicine, M.L.B. Medical College, Hospital, Jhansi.

SELECTION OF CASES

The study comprised of pregnant females admitted to the antenatal clinic, labour room, and maternity wards during antenatal or various stages of labour. A total of 102 cases were considered and of these only 52 cases were taken for final assessment. Rest of the patients were excluded from the study because their blood samples were discoarded due to some problems like short hospital stay and hemolysis etc. Patients of high risk group were also excluded from the study. cases selected for the study were free from any complications of pregnancy.

METHOD

After selection of cases a complete detailed history of patients was taken with emphasis on age, address, socio-economic status and occupation.

Obstetric History

- Date of last menstrual period or duration of amenorrhoea.
- Gravida and parity.

- Previous still birth.
- Duration of onset of pain
- Any accompaniments i.e. leaking or bleeding per vaginum.

Past History

Any past history of pregnancy induced hypertension, eclampsia, intrauterine growth retardation and pre & post maturity, medical disorders like hypertension, diabetes, tuberculosis, renal and liver diseases and thyroid disorders were excluded.

Family History

Diseases like coronary heart disease, hypertension, and diabetes were excluded in the family of the patients.

Personal History

Tobacco chewing, alcohol intake, smoking and therapy with corticosteroid, thiazides, betablockers with special emphasis on hormonal contraceptives were excluded.

Dietary History

Daily intake of food with emphasis on fat and calories per day, type of diet whether vegetarian or non vegetarian was assessed.

EXAMINATION : General examination

Detailed examination with special emphasis upon built, weight (serial measurement), pulse, blood pressure, pallor, icterus and oedema, was done.

Obstetric Examination

1. Per abdominal Examination (P/A) :
   - Assessment of gestational age by final height.
   - Lie and presentation of fetus.
   - Fetal heart sound and amount of liquor adequate or not.

2. Per vaginal examination (P/V) : To know -
   - Dilation of cervix.
   - Thinning of cervix.
   - Presenting part and its station.
   - Colour of liquor to know the condition of fetus. whether normal or in distress.
   - To decide the stage of labour for withdrawing the blood samples at proper time for our study.
   - To assess the pelvis whether it is adequate for normal vaginal delivery or not.

INVESTIGATIONS

Blood : Haemoglobin
Blood group
Blood sugar (Fasting and post prandial)
V.D.R.L. to exclude syphilis.

Urine : Routine - Albumin

Sugar

Microscopic examination.

Procedure for blood sampling

3-4 ml blood from antecubital vein with minimal stasis on recumbent posture was withdrawn under aseptic conditions. Samples were collected in autoclaved vials and allowed to settle for half an hour to separate the serum. Further procedure and estimation of lipoproteins was done at the Lipid Research Laboratory, Department of Medicine, M.L.B. Medical College, Jhansi.

Blood samples were collected during the process of labour or was contemplated to start within a short period. Following blood samples were taken :

1. In early labour. - at the onset.
2. End of 1st stage - full dilatation of cervix.
3. End of 2nd stage - after delivery of baby but before expulsion of placenta.
4. End of 3rd stage - after delivery of placenta.
5. Blood sample with 24 hours of post partum.
6. Blood from cord of placental side.

The above samples were estimated for serum total cholesterol (STC), serum triglycerides (STG), and high density lipoprotein (HDL) by the standard methods. Very low density lipoprotein (VLDL) and low density lipoprotein (LDL) were calculated by the following formulae :

VLDL (mg/dl) = STG/5 (Valid upto the values of STG ⩽300 mg/dl).

LDL (mg/dl) = STC - (STG/5 + HDL)

STC - (VLDL + HDL)

For the study, patients selected, were divided into following groups according to mode of delivery :

Group I : Subjects having normal spontaneous vaginal delivery served as control group.

Group II : Subjects having vaginal delivery where artificial rupture of membrane (A.R.M.) was done to induce labour.

Group III : Subjects with normal vaginal delivery where artificial rupture of membrane and oxytocin augmentation was done.

Group IV : Normal subjects who underwent elective caesarean section.

Group V : Normal subjects who underwent emergency caesarean section due to obstructed labour.

Lipoprotein profile between these 5 groups were compared during 1st, 2nd, 3rd stage of labour and within 24 hours of postpartum period.

It was also tried to know the effect of parity on lipoprotein profile. Subjects were divided according to parity - primigravida and multigravida. The lipoprotein profile were compared in these two groups during intrapartum and 24 hours postpartum period.

Umbilical cord blood of newborns from placental side was studied for lipoprotein profile changes in relation to mode of delivery and diet - vegetarian and non-vegetarian.

O B S E R V A T I O N S

OBSERVATIONS

In the present study 52 cases were taken for the final analysis. General characteristics of the cases were as follows :

Age :	Range	18-35 years
	18-25 years	35(67.31%)
	25 years	17(32.69%)
Weight -	Mean±S.D.	54±7 kg.
Parity :	Primipara	11(21.15%)
	Multipara	41(78.85%)
Socio-economic status		
	Low	30(57.59%)
	Middle	22(42.41%)
Dietary habit :		
	Vegetarians	27(51.92%)
	Nonvegetarians	25(48.08%)

None of the subject was habituated to smoking, alcohol or tobacco. None was taking oral contraceptive and had not any medical disorders. The subjects of caesarean section were on intravenous fluids.

Study was carried out during 1st, 2nd and 3rd stage of labour and within 24 hours of postpartum period.

For the study, total 52 subjects selected for final assessment were divided into following groups according to mode of delivery.

Group I

This group included 18 cases having normal spontaneous vaginal delivery and served as control group.

Group II

This group consisted of 5 patients having vaginal delivery where artificial rupture of membrane was done to induce labour.

Group III

In this group, 6 subjects with normal vaginal delivery where artificial rupture of membrane and oxytocin augmention was done, were included.

Group IV

Ten subjects who underwent elective caesarean section, were put in group IV.

Group V

This group consisted of 10 normal subjects who underwent emergency caesarean section due to obstructed labour.

TABLE I : STC levels during various stages of labour and 24 hours postpartum period in different groups (Mean±S.D., mg/dl).

Groups	Stage I	Stage II	Stage III	Postpartum period
Group I	198.87 ±23.78	196.33 ±17.46	188.83 ±17.48	164.61 ±19.99
Group II	196.20 ±15.42	191.40 ±14.40	185.20 ±13.70	167.80 ±11.99
Group III	200.17 ± 9.50	195.00 ± 8.83	186.69 ± 7.09	174.33 ± 6.62
Group IV	-	-	184.40 ±14.86	170.90 ±19.91
Group V	-	155.63 ±16.58	170.09 ±17.66	160.07 ±19.66

Table I shows that STC levels fell from stage I to stage III and there was further fall in postpartum period in all the groups except in group V. In group V cases there was rise in STC levels from stage II to III of intrapartum period and a fall in postpartum level when compared with intrapartum levels.

STATISTICAL ANALYSIS

In group V	't'	'p'
Stage II Vs III	6.73	< 0.001
Stage II Vs PP	5.92	< 0.001

	Group I Vs II		Group I Vs III	
	't'	'p'	't'	'p'
Stage I	0.26	>0.05	0.18	>0.05
Stage II	0.59	>0.05	0.23	>0.05
Stage III	0.16	>0.05	0.54	>0.05
PP	0.41	>0.05	1.71	>0.05

	Group I Vs IV		Group I Vs V	
	't'	'p'	't'	'p'
Stage I	-	-	-	-
Stage II	-	-	6.63	<0.001
Stage III	0.09	>0.05	2.07	<0.05
PP	0.80	>0.05	0.61	>0.05

Statistical analysis shows that from group I to IV there was insignificant fall during various stages of labour and a significant fall in postpartum values of STC.

In group V there was significant rise from stage II to stage III and a significant fall in postpartum period when compared with values of intrapartum period.

Statistical analysis also shows that when group I was compared with groups II, III and IV there was no any significant difference in STC levels in ant stage of labour and postpartum period. While in group V there were significantly low values as compared to group I during various stages of labour.

During plstpartum phase, there was no significant change in STC values in group V when compared with group I postpartum period values.

TABLE II : Serum triglycerides levels during various stages of labour and 24 hours postpartum periods in different groups of subjects. (Mean±S.D., mg/dl).

Groups	Stage I	Stage II	Stage III	Postpartum period
Group I	118.83 ±18.20	112.78 ±17.65	103.94 ±16.97	94.56 ±16.87
Group II	114.40 ±16.92	107.60 ±17.29	97.00 ±19.66	90.60 ±18.84
Group III	113.00 ±16.14	106.33 ±14.40	100.50 ±15.86	92.83 ±13.82
Group IV	-	-	107.60 ±12.97	97.20 ± 9.75
Group V	-	108.39 ±14.30	104.39 ±14.07	94.31 ±11.74

Table II shows that there is fall in STG values in all groups during intrapartum period and postpartum period.

Statistically the fall during intrapartum period was insignificant while there was significant fall in post-partum STG values when compared with intrapartum period. values.

STATISTICAL ANALYSIS

Intrapartum Vs Postpartum :

	't'	'p'
Group I	2.20	<0.05
Group II	2.99	<0.05
Group III	3.03	<0.05
Group IV	2.94	<0.05
Group V	2.59	<0.05

	Group I Vs II		Group I Vs III	
	't'	'p'	't'	'p'
Stage I	0.49	70.05	0.70	70.05
Stage II	0.58	70.05	0.81	70.05
Stage III	0.78	70.05	0.44	70.05
PP	0.45	70.05	0.23	70.05
	Group I Vs IV		Group I Vs V	
	't'	'p'	't'	'p'
Stage I	-	-	-	-
Stage II	-	-	0.74	70.05
Stage III	0.59	70.05	0.14	70.05
PP	0.45	70.05	0.05	70.05

There was no alteration in values of STG between the groups. This shows that the mode of delivery did not produce any significant change during intrapartum and postpartum periods.

TABLE III : HDL levels during various stages of labour and 24 hours postpartum period in different groups (Mean±S.D., mg/dl).

Groups	Stage I	Stage II	Stage III	Postpartum period
Group I	54.00 ±16.18	49.33 ±14.66	48.72 ±11.69	46.74 ±10.85
Group II	53.00 ± 9.85	51.60 ± 6.35	44.40 ± 6.99	37.40 ± 7.36
Group III	49.50 ±11.15	46.17 ±11.75	41.00 ± 9.65	35.67 ± 8.98
Group IV	-	-	51.90 ± 8.60	39.40 ± 6.69
Group V	-	47.08 ±10.40	46.62 ± 9.64	45.39 ± 5.10

Table III shows that there was fall in HDL levels in intrapartum period and there was a further fall in post partum period when these levels were compared with that of intrapartum period.

On statistical analysis it was seen that there was no significant fall during intrapartum phase in any stage of labour but when compared with postparym period there was significant fall in HDL levels of all the five groups.

Statistical analysis between stages

Groups	Stage I Vs II		Stage I Vs III		Intrapartum Vs postpartum	
	't'	'p'	't'	'p'	't'	'p'
Group I	1.05	>0.05	2.01	>0.05	3.06	<0.01
Group II	0.73	>0.05	1.09	>0.05	3.44	<0.05
Group III	0.91	>0.05	1.63	>0.05	2.70	<0.05
Group IV	-	-	-	-	2.49	<0.05
Group V	-	-	1.08	>0.05	2.97	<0.05

Statistical analysis between groups

Stages	Group I Vs II		Group I Vs III	
	't'	'p'	't'	'p'
Stage I	0.13	>0.05	0.63	>0.05
Stage II	0.34	>0.05	0.48	>.05
Stage III	1.38	>0.05	1.69	>0.05
PP	1.80	>0.05	1.85	>0.05
	Group I Vs IV		Group I Vs V	
	't'	'p'	't'	'p'
Stage I	-	-	-	-
Stage II	-	-	0.48	>0.05
Stage III	1.10	>0.05	0.53	>0.05
Stage - PP	1.82	>0.05	0.59	>0.05

Statistical analysis shows that there was no significant change in various groups during stages of labour and postpartum period in HDL Values.

TABLE IV : LDL levels during various stages of labour and 24 hours postpartum period in different groups (Mean±S.D. mg/dl).

Groups	Stage I	Stage II	Stage III	Postpartum period
Group I	117.61 ±14.69	114.44 ±13.46	113.28 ±12.14	99.09 ±12.11
Group II	120.20 ±15.47	118.20 ±14.75	116.20 ±11.59	112.40 ± 9.47
Group III	128.17 ±10.68	127.50 ±10.41	120.50 ± 9.16	113.17 ± 8.26
Group IV	-	-	108.00 ± 9.16	107.90 ± 8.25
Group V	-	86.86 ± 6.59	105.55 ± 5.37	85.40 ± 4.76

Table IV shows that from stage I to III of intrapartum period there was a fall and a further fall in postpartum period levels of LDL when compared with intrapartum levels from group I to IV.

In group V there was rise from stage II to III LDL levels a fall in postpartum values when compared with stage II and III of labour.

Statistical Analysis

	Stage II Vs III		Stage II Vs PP	
	't'	'p'	't'	'p'
Group I	0.87	>0.05	3.09	>0.05
Group V	3.43	<0.01	2.13	<0.05

	Group I Vs II		Group I Vs III	
	't'	'p'	't'	'p'
Stage I	0.35	70.05	1.61	70.05
Stage II	0.57	70.05	2.06	70.05
Stage III	0.49	70.05	1.59	70.05
PP	2.01	70.05	2.06	70.05

	Group I Vs IV		Group I Vs V	
	't'	'p'	't'	'p'
Stage I	-	-	6.81	⟨0.001
Stage II	-	-	2.29	⟨0.05
Stage III	1.16	70.05	2.045	⟨0.05
PP	2.04	70.05	-	-

Statistical analysis shows that there was an insignificant fall during stage I to III of intrapartum phase and a significant fall in postpartum period in the group I to IV.

There was a significant rise from stage II to III of intrapartum period and a significant fall during 24 hours postpartum in group V.

On comparing group I to II, III and IV, there was no significant difference in values during intrapartum and postpartum period while on comparing the values of group V with group I there were significantly low values during stage II and III of labour and an insignificant fall in postpartum phase in group V.

TABLE V : VLDL values during various stages of labour and 24 hours postpartum period in different groups (Mean±S.D., mg/dl).

Groups	Stage I	Stage II	Stage III	Postpartum period
Group I	23.76 ±3.63	22.56 ±3.55	21.83 ±3.38	18.89 ±3.36
Group II	23.00 ±3.30	21.60 ±3.36	19.60 ±4.04	18.12 ±3.54
Group III	22.50 ±3.21	21.33 ±2.80	20.17 ±3.31	18.50 ±2.88
Group IV	-	-	21.50 ±2.51	19.60 ±1.96
Group V	-	21.69 ±2.75	20.58 ±2.78	19.34 ±2.53

Tavle V shows that there was a fall in VLDL values during stage I, II, III of labour and a further fall in postpartum period in all groups.

STATISTICAL ANALYSIS

Intrapartum Vs postpartum

	't'	'p'
Group I	3.63	<0.01
Group II	2.86	<0.05
Group III	2.75	<0.05
Group IV	2.64	<0.05
Group V	2.53	<0.05

	Group I Vs II		Group I Vs III	
	't'	'p'	't'	'p'
Stage I	0.40	>0.05	0.73	>0.05
Stage II	0.54	>0.05	0.76	>0.05
Stage III	0.78	>0.05	0.47	>0.05
PP	0.52	>0.05	0.25	>0.05

	Group I Vs IV		Group I Vs V	
	't'	'p'	't'	'p'
Stage I	-	-	-	-
Stage II	-	-	0.73	>0.05
Stage III	0.58	>0.05	0.12	>0.05
PP	0.61	>0.06	0.001	>0.05

Statistical analysis shows that there was an insignificant fall in VLDL levels during stages of labour in all groups while there was a significant fall in VLDL values during postpartum period when compared with intra-partum values. Analysis also shows that mode of delivery does not show any significant change in values from stage I to III and postparum period.

Table VI : Various lipoprotein fractions in umbilical cord blood of newborns of different groups (Mean±S.D., mg/dl).

Groups	STC	STG	HDL	LDL	VLDL
I	74.22 ±11.49	67.64 ±6.202	34.28 ±2.61	26.26 ±8.59	13.68 ±1.23
II	80.00 ±8.86	65.80 ±5.01	33.60 ±3.44	33.24 ±6.37	13.16 ±0.88
III	79.33 ±7.80	70.33 ±7.65	33.33 ±2.16	31.94 ±5.03	14.06 ±1.39
IV	80.80 ±8.12	67.72 ±4.26	36.00 ±3.40	31.07 ±4.96	13.73 ±4.12
V	73.77 ±10.50	69.00 ±6.40	32.72 ±3.15	37.05 ±3.04	18.80 ±1.28

Statistical Analysis

Lipid lipoprotein	Groups							
	I Vs II		I Vs III		I Vs IV		I Vs V	
	't'	'p'	't'	'p'	't'	'p'	't'	'p'
STC	1.04	70.05	1.01	70.05	1.60	70.05	1.10	70.05
STG	0.61	70.05	0.87	70.05	0.04	70.05	0.60	70.05
HDL	0.48	70.05	0.80	70.05	1.50	70.05	1.30	70.05
LDL	1.68	70.05	1.52	70.05	1.62	70.05	0.32	70.05
VLDL	0.88	70.05	0.63	70.05	0.11	70.05	0.26	70.05

Statistical analysis of Table VI shows that there was no any significant difference in values of various lipoprotein fractions in all five groups i.e. mode of delivery does not affect the cord blood values of newborns.

TABLE VII : Showing lipoprotein fractions in relation to parity during intrapartum and postpartum periods (Mean±S.D., mg/dl).

Lipoproteines		Intrapartum	Postpartum
STC	Primipara	192.72±16.51	174.72±21.63
	Multipara	184.22±15.07	169.43±17.21
	't'	1.63	0.86
	'p'	70.05	70.05
STG	Primipara	104.00±16.49	95.45±94.14
	Multipara	103.73±15.26	94.14±13.89
	't'	0.0509	0.274
	'p'	70.05	70.05
HDL	Primipara	46.909±8.28	35.09±5.12
	Multipara	42.759±11.18	37.56±6.20
	't'	11.18	1.21
	'p'	70.05	70.05

Table VII shows that numerically the lipoprotein fractions levels were higher in primipara as compared to multipara during intrapartum and postpartum periods. Statistical analysis shows that there was no any significant difference in lipoprotein fractions between primipara and multipara during intrapartum and postpartum periods.

TABLE VIII : Showing lipoprotein fractions values during intrapartum, postpartum periods and umbilical cord blood of newborn in relation to diet of mother (vegetarian Vs non-vegetarian) (Mean±S.D., mg/dl).

Lipoproteines		Intrapartum	Postpartum	Cord blood
STC :	Vegetarian	185.925 ±14.681	170.00 ±17.47	73.52 ±11.45
	Nonvegetarian	186.12 ±16.89	171.16 ±19.16	76.26 ±10.18
	't'	0.044	0.228	0.90
	'p'	>0.05	>0.05	>0.05
STG :	Vegetarian	102.3704 ±17.81	92.81 ±16.02	78.78 ±10.89
	Nonvegetarian	105.32 ±12.83	96.15 ±11.32	77.84 ±9.12
	't'	0.688	0.86	0.34
	'p'	>0.05	>0.05	>0.05
HDL :	Vegetarian	46.41 ±10.87	37.96 ± 6.41	33.70 ±3.11
	Vegetarian	45.44 ±10.67	36.04 ± 5.55	34.48 ±2.96
	't'	0.32	1.15	0.88
	'p'	>0.05	>0.05	>0.05

On statistical analysis there was no any significant changes in values of lipoprotein fractions in vegetaria Vs non.vegetarian mothers when blood was withdrawn during intrapartum, postpartum and cord blood samples of newborns.

D I S C U S S I O N

D I S C U S S I O N

The present study was conducted to know the lipoprotein profile changes during the process of labour and 24 hours of postpartum period in relation to mode of delivery, parity, diet etc in various groups of cases.

Of the total 102 cases, only 52 were considered for final assessement, rest of the patients were excluded from the study due to their blood samples were not sufficient for study and further followup of patients. was not possible. patients studied were normal individuals with no any cardiac problem or any other medical or obstetrical complication and were of average built and weight. No one was grossly obesed.

For the study subjectsewere catagorised into five groups according to their mode of delivery. Normal healthy pregnant females who delivered spontaneously served as contro and were compared to subjects delivered by induction either b artificial rupture of membrane. ARM + oxytocin infusion and elective or emergency caesarean section. In cases of elective caesarean section there was no sample of stage I and II while in emergency caesarean section due to obstructed labou stage I sample were not available.

The following pattern of lipoprotein profile change were found during intrapartum and postpartum periods.

SERUM TOTAL CHOLESTEROL (STC)

The values of STC in control group were 198.87± 23. mg/dl during stage I am sere decreased to 183.83±17.48 mg/dl at stage III of labour.

The values were tend to fall with same pattern in induced delivery and in elective caesarean section cases as in control group. with insignificant fall (p 70.05).

Subjects of group V i.e. cases of obstructed labour who had emergency caesarean section were presented with the picture different from that of other groups. In this group STC levels were significantly lower than any other group and were tend to increase from the time of admission. to stage III i.e. 155.63±16.58 to 170.09±17.66 mg/dl (p $\leq$ 0.001).

This dissimilar pattern in cases of obstructed labour is not due to stress, the possible mechanism is that the regulatory mechanism in the body ensure a supply of adequate fuel for all the cells of body whenever required, in totally starvated condition of patients or fully fed state. This mechanism is disturbed during pregnancy due to hormonal imbalance. Thus in cases of obstructed labour due to starvated and dehydrated, condition there was fall in blood glucose level and decreased glucose availability, so low insulin hormone level in blood and also there was decrease in HMGCOA activity. Thus, serum cholesterol synthesis is difficult. also reduced insulin levels lead to increase in lipolysis and so there is increase in free fatty acid levels. When insulin level is low, it disturbs the antilipolytic activity of PGE (the level of PGE are maximum during labour). and thus, there was further increase in lipolysis. In cases of obstructed labour due to contraction of uterus, continuously with pain and dehydrated state there is loss of glucose as all the glucose is consumed by contracted uterus. It is known

that in fed state, the free fatty acid are esterified into triglycerides but in case of obstructed labour patient is dehydrated and had low blood glucose level, thus free fatty acids instead of their esterification are oxidised and for this reason STG level becose low. As from table I it is clear that there was rise in STC values gradually from initial to III stage of labour in grou V. It is presumed that increased values of STC in later stages is due to intravenous line (10% glucose, 25% glucose etc). having adequate amount of glucose were provided to obstructed labour cases. This glucose promotes esterification of fatty acids and also initiated lipolysis through PGE activation and thus there was raise in STC in group V.

In postpartum phase, there was significant fall in STC values in all five groups within 24 hours e.g. in control group, values were 198.87_23.78 mg/dl in stage I of intrapartum period to fall in 24 hour postpartum (164.61_19.99 mg/dl).

Pother and Nestel (1979) noted the fall of 14% in STC value s within 24 hours of delivery.

Arora and Kavita et all (1989) showed that in normal and troxaemia group of patients STC levels fell just after delivery.

Another study which is going parallel to present study (Arora and Majeed, 1998) showed that the STC values during intrapartum period was 243.07± 25.72 mg/dl and declined to 222.10±26.52 mg/dl in 24 hours of postpartum.

It is known that lipoprotein fractional changes during pregnancy were affected by placental hormones e.g. oestriol, conjugated oestragen human placental lactogen,

Oestriol is main pregnancy oestrogen accounts for 80-90% of oestrogen formed in last trimester.

According to Eliert (1949) administration of cestrogen to women evoked on increase in plasma lipid level, according to Kilopper (1978) by 8 hours of postpartum phase the total oestreiol levels fall to 33%, Unconjugated oestriol levels fall to 20%. of its basal intrapartum values. HPL falls steeply by 3 hours. Urinary cestrogen at term is 40-50 mg/24 hours urine and less than 12 mg/24 hours urine indicates serious fetal compromise in utero during later month of pregnancy.

Thus it can be said that fall in postpartum STC values were due to fall in placental hormone level.

SERUM TRIGLYCERIDE : (STG)

In present study, the STG levels showed decreasing trend from stage I to III of intrapartum phase in all the groups. This decrease was insignificant ($p > 0.05$). The STG levels of control group were 118.00±18.20 mg/dl 112.78±17.65 and 103.94±16.97 mg/dl during I, II and III stage of labour respectively.

Previous study by neeta et al (1993) explained that higher STG levels during intrapartum phase in cases of emergency caesarean section is due to prolonged obstructed labours and stress of poor fetal outcome but in present study the values obtained were not higher than other group of patients i.e. control, induced and elective caesarean section cases. i.e. 108.38±34.30 mg/dl to 104.77 ±14.07 mg/dl during intrapartum phase of emergency caesarean section due to obstructed labour cases.

Pontis et al (1997) explained that labour is a stressful condition which leads to catecholamine release and these catecholamines alter the estrogen:progesterone ratio and the release of prostaglandian E2 which is anti lipolytic by action and thus free fatty acids are estirified to triglycerides and so these is an increase in Serum triglyceride levels in obstructed labour cases, but in present study the STG levels were within the limit when compared to other groups.

In postpartum phase there was a fall in STG levels in all the five groups. this fall in control group was 118.83±18.20 mg/dl to 94.56±16.87 mg/dl during 24 hours of postpartum phase as compared to intrapartum values. This fall was significant ($p < 0.05$) in all the groups irrespective of mode of delivery.

Watson (1957), De Alverez et al (1959), Konttinen et al (1964) have shown a fall in STG level during puerperium.

Potter and Nestel (1979) showed fall of 24% in plasma triglyceride levels within 24 hours of delivery.

Thus, it can be said that the fall in STG is statistically significant in 24 hours of postpartum period and this is due to lack of placental hormones. there is no difference of time of falling values of STG by any mode of delivery in present study.

HDL (HIGH DENSITY LIPOPROTEIN)

There was no any significant change in HDL levels during intrapartum phase while there is significant fall in postpartum values. The control group showed

HDL level s an 54.00±16.18, 49.33±14.46, 48.72±11.69 and 46.74±10.85 mg/dl at I, II, III stage of intrapartum period and 24 hours of postpartum period respectively. There was no intergroup variation of intrapartum levels. But postpartum values in all groups had significant variation when compared with intrapartum values of the same group. These changes were probably due to falling placental hormone levels. As it is known that estrogen increases the HDL levels and estrogen levels decreases during postpartum period thus there was a fall in HDL levels also.

LOW DENSITY LIPOPROTEIN (LDL)

LDL levels showed same trend as that of STG. The values of LDL in control group were 117.61±14.69, 114.94±13.96, 113.28±12.14 mg/dl during I, II and III stage of labour that is decreasing from stage I to III but values were insignificant and no intergroup significant was present.

In group V, LDL levels were low and significant as compared to control group. These LDL levels tend to increase from time of admission to stage III of lavour i.e. 86.86±6.59, 105.55±15.37 mg/dl and the increase was significant ($p<0.01$). The LDL level showed similar pattern of variation as that of STC in postpartum phase. The values decreased from 117.61±15.69 to 99.09±12.11 mg/dl within 24 hours post partum pieiod in control group ($p<0.05$). Similarly in all other groups same pattern followed significantly irrespective of mode of delivery.

VERY LOW DENSITY LIPOPROTEIN (VLDL)

It is known that VLDL is derived from STG, (VLDL= STG/5), thus it showed the parallel relationship with STG. The values of FLDL fall from I to III stage of labour i.e. 23.76±3.63, 22.56±3.55 and 21.83± 3.38 mg/dl respectively in control group.

This fall was insignificant in all the groups during intrapartum phase but there was significant fall during postpartum phase i.e. 23.76±3.63 to 18.89±3.36 mg/dl. The reason may be same as that of fall in STG.

LIPOPROTEIN FRACTIONS IN CORD BLOOD

Cord blood levels of lipoprotein fractions were studied in all the five groups. It showed that the levels of all lipoprotein fractions were low as compared to maternal blood values. The lipoprotein fractions among the groups did not showed any significant difference i.e. with mode of delivery.

Broady & Carlson (1962) found that the concertration of STG was quite low in cord blood and it was confirmed by Kaplan and Levi (1965). It is clear from previous studies that in cases where baby undergoes stress, in vitro, there is increase in triglyceride levels.

EFFECT OF PARITY

Lipoprotein changes in relation to parity were compared during intrapartum and postpartum period in

Table VII. Numerically the lipoprotein fraction levels were higher in primipara as compared to multipara during intrapartum and postpartum period but the differences were insignificant.

EFFECT OF DIET

There was no any significant variation in values of lipoprotein fractions in vegetarian Vs nonvegetarians subjects during intrapartum , postpartum period and also incord blood of new borns.

Arora and Vinita (1987) studied the influence of dietary fat on STC level during anti partum, intrapartum and pospartum phase and in cord blood of newborns. They concluded that the levels of STC were higher in subjects taking high fat diet and lower in subjects taking normal or low fat diet. With advancement of pregnancy, during labour and after delivery, values were not statistically significant.

SUMMARY AND CONCLUSION

SUMMARY AND CONCLUSION

The present study was conducted to know the lipoprotein profile changes during the process of labour and 24 hours of postpartum period and also to know the changes in lipoprotein pattern in relation to mode of delivery (i.e. normal vaginal delivery without any intervention, induced delivery and caesarean section (elective or emergency) parity and diet etc various variable.

52 cases including 11 primigravidae, and 41 multigravidae in age group of 18-35 years and mean weight of 54±07 kg were studied during intrapartum and post partum phase. All cases were categorised according to mode of delivery into five groups.

Group I comprised the cases of spontaneous veginal delivery and served as control group. Group II comprised the cases in those artificial rupture of memberane was done to induce labour. Group III included the cases in those A.R.M. and oxytocin infusion both were used to augment labour. Group IV included the cases in those elective caesarean section was done, and cases of due to obstructed labour emergency caesarean section were put in group V.

All the five groups of cases were studied during various stages of labour and 24 hours postpartum to detect whether there is any significance of diet on lipoprotein profile changes or not and also was there any difference in values of liprotien fractions in between primi and multi gravidae.

Umbilical cord blood from placental side was withdrawn for the study of various lipoprotein fractions in all the five groups i.e. to study the changes in lipoprotein fractions of umbilical cord blood according to mode of delivery. After the whole study , in summary it can be said that lipoprotein profile changes during intrapartum and within 24 hours of postpartum periods showed following pattern.

The STC values decreases during intrapartum period from one to another stage of labour in patients of normal and induced vaginal deliveries while in cases of emergency caesarean section due to obstructed labour there was an increase in values of STC, during intrapartum period. This change of increasing values in cases of groups V was significant while decrease in valu[illegible] of STC during intrapartum period in rest of the groups were insignificant.

During post partum period there was significant fall irrespective of mode of delivery in all five groups.

LDL values followed the same pattern as that of STC.

STG levels were decreased during intrapartum period from one to another stage of labour in all the five groups. during post partum period, There was further fall in values of STC when compared with that of intrapartum values and are significant statistically. HDL and VLDL values showed the same pattern as that of STG. On comparing the various lipoprotecin fractions in the umbilical cord blood of newborns borned by different modes of delivery. There was no any significant difference according to the mode of delivery of mother i.e. lipoprotein fractions values of umbilical cord blood of newborn does not depends on their mode of expulsion.

To note the effect of parity on lipoprotein profile the cases were divided into two groups i.e. primigravidae (11 cases) and multigravide (41 cases). The lipoprotein changes showed the similar changes in either groups during intrapartum and post partum periods, the intergroup difference was statistically insignificant.

To, detect the effect of diet on lipoprotein profile the cases were divided into vegetarian and non-vegetarian categories. There were 27 vegetarian and 25 nonvegetarian cases in present study. Effect of diet on cord blood lipoprotein fractions in various groups of cases was also studied. It was found that there was similar trend in lipoprotein fractional values in both vegetarian and nonvegetarian cases i.e. no any significant

difference was found between the two categories of cases irrespective of mode of delivery. Similarly there was not significant difference in umbilical cord values of lipoprotein in vegetarian and nonvegetarian cases.

To conclude, there was a statistically insignificant fall in STC, LDL values during intrapartum period in cases of spontaneous delivery or induced and elective casearean section. In cases of emergency caesarean section due to obstructed labour there was significant increase in these values during intrapartum phase. During post partum phase there was insignificant fall in all five groups of cases.

In case of STG, HDL and VLDL there was fall in intrapartum values during various stages in all the groups but the fall was insignificant while there was a significant fall during postpartum period.

The values of lipoprotein fractions were not affected by diet, parity and mode of delivery etc. Umbilical cord blood lipoprotein values also did not show any significant relation with mode of delivery, diet etc. Thus, it can be said that the changes onserved were normal physiological phenomenon.

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